CHAPTER 6
Chest Pain

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The challenge in accurately diagnosing chest pain arises from several important neuroanatomic facts. First, neither the quality nor the intensity of pain produced by the nerves coming from the thoracic viscera is specific for any single organ system. Spasm of the esophagus, ischemia of the heart, or distention of the great vessels can produce feelings of pressure, aching, or burning. Pain can be severe, minimal, or even absent during the course of life-threatening conditions such as an acute myocardial infarction.

Second, the location and radiation of the pain do not reliably identify the specific organ system involved. Thoracic organ pathology can produce pain that is referred outside the thorax to the epigastrium, neck, or jaw. Conversely, cervical pathology from a ruptured cervical disk can produce pain in the shoulder and around the clavicle. Abdominal pathology, such as a ruptured ectopic pregnancy, can produce chest and scapular pain due to diaphragmatic irritation.

The anatomic explanation for referred pain is simple. Somatic afferent nerves from the skin and muscle of the arms enter the same dorsal root nerve pools as the visceral afferent nerves from the heart, esophagus, and other thoracic organs. Activation of this nerve pool by visceral afferents can stimulate the somatic afferent nerves. The brain interprets the pain as coming from the arm, muscle, or joint innervated by the pooled somatic afferents. Pain from the heart can be perceived as pain in the upper inner arm, forearm, or axilla, because both areas synapse in the dorsal roots T1–T5 (10). Also, because dorsal nerve segments overlap, three segments above and below a particular level, thoracic pain can be referred to the neck or abdomen. Therefore, to identify the organ system and the disease process, the physician must rely on the duration and frequency of the pain, the setting in which it occurs, the aggravating and relieving factors, and associated symptoms.

The five major thoracic organ systems are listed in Table 6.1. The most common etiologies that affect these organs can be categorized as structural disruption, infection or ischemia, infection, and inflammation.

CLINICAL PRESENTATION

The historical features of chest pain are useful in establishing the “pretest” probability for each disease in the differential diagnosis. Such probabilities guide the choice and interpretation of further diagnostic tests.

Acute myocardial infarction (AMI) produces a nonlocalized pressure, ache, or burning, the intensity of which ranges from minimal discomfort to severe pain. The pain is usually substernal and in the left chest, but can occur between the umbilicus and the neck. It frequently radiates to the shoulder or left arm and lasts several hours.

Stuttering presentations may last 12 to 24 hours. Determining the onset of constant pain is essential in deciding whether the patient is eligible for acute reperfusion therapy. Dyspnea, diaphoresis, nausea, and weakness are frequently associated symptoms. Occasionally, pain is felt only in a referred area, such as the arm, or via the vagus nerve, in the ear. Patients with AML particularly the elderly and diabetic patients, often present without pain (1), and this diagnosis must be suspected with the presentation of syncope or confusion.

Pain features not suggestive of AMI include stabbing, knife-like sensations or radiation to areas outside of cervicothoracic nerve segments, such as the legs or flanks (6). Very brief pain (lasting less than 5 seconds) or pain that is clearly pleuritic or exactly reproduced by bending or palpation is very unlikely to be of coronary origin (12).

The pain of angina is similar to that of AMI but is of shorter duration. A clear exertional pattern is helpful to the diagnosis; pain can also be provoked by effort, emotion, or exposure to cold. Pain relief within 5 minutes from sublingual nitroglycerin is suggestive of angina but can also occur with esophageal spasm or placebo. Pain that is relieved with exertion and brought on by rest is not anginal. Angina is unstable when it increases in severity (duration, intensity, frequency), occurs with reduced activity or at rest, or has been present for less than 4 weeks. This diagnosis requires hospital admission or risk stratification prior to discharge from the ED.

Aortic dissection classically produces a severe tearing pain in the anterior chest radiating to the back, flank, or arm. The patient sometimes feels pain traveling down the back or flank as the dissection extends distally. The pain is frequently described as being migratory. Its onset is sudden, but it may be intermittent or wax and wane. Uncommonly, it presents as a myocardial infarction or as a chronic pain that has become worse. The pain has no relieving factors, and withing, diaphoresis, dyspnea, nausea, and vomiting are commonly associated. A common error is to dismiss the diagnosis of aortic dissection because the pain gets better with nitroglycerin. This may be due to a decrease in the intraluminal pressure of the aorta after nitroglycerin. Associated symptoms, such as weakness, paralysis, syncope, and tinnitus or pain in an extremity, are also common and more specific for dissection.

Pericarditis produces a sharp or aching pain in the precordium that may radiate to the scapula, neck, or shoulder. Unlike AMI, it has a long duration (days), and can be continuous and severe or pleuritic. It is usually made worse by lying down or breathing; thus, the associated “shortness of breath.” Patients may insist on leaning forward to make themselves more comfortable.

Myocarditis can present with associated pericarditis. It also uncommonly masquerades as a myocardial infarction (13). Antecedent viral illness and younger age are diagnostic clues, but an in-hospital evaluation is necessary for definitive diagnosis of this uncommon cause of chest pain.

The most common presentation of pulmonary embolism (PE) is dyspnea, pleuritic pain, or hemoptysis (20). About 85% of cases of PE present with one or more of these nonspecific symptoms. Tachypnea is common, but tachycardia is found in only a minority of proven cases of PE. Large pulmonary emboli can produce circulatory collapse with syncope due to acute pulmonary hypertension and right heart failure. PE is mistaken for AMI about 7% of the time (20). Chest pain that is strictly reproducible is not consistent with PE, but rarely is present in these patients.

Pneumothorax may produce a severe, sudden stabbing pain in the affected side, or it may be asymptomatic. It has no characteristic pain radiation. It is made worse by breathing, is relieved by splinting, and is associated with shortness of breath and nonproductive cough. Pleurisy, an inflammation of the parietal pleura, is similar but does not have the associated symptoms or radiographic findings. Pneumonia can also cause pleuritic pain, but its association with productive cough, shortness of breath (at rest or exertion), and fever is often a helpful differentiating feature.

Pain may be due to an esophageal source. Pain from esophageal spasm is often indistinguishable from angina in quality, intensity, location, and radiation. Spasm has been described as a perfect mimic of acute myocardial ischemia. Esophageal reflux is a
<table>
<thead>
<tr>
<th>Organ System/Etiology</th>
<th>Chest Pain</th>
<th>Associated Symptoms</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>CARDIOVASCULAR</strong></td>
<td></td>
<td></td>
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<tr>
<td>Acute myocardial infarction</td>
<td>Pressure, aching, burning</td>
<td>Dyspnea, palpitations, nausea, diaphoresis, radiation</td>
</tr>
<tr>
<td>Aortic dissection</td>
<td>Sudden, severe, tearing</td>
<td>Back pain, neurovascular deficits</td>
</tr>
<tr>
<td>Unstable angina</td>
<td>Same as AMI except episodic</td>
<td>Same as AMI</td>
</tr>
<tr>
<td>Pericarditis</td>
<td>Sharp, pleuritic, positional</td>
<td>Fever, dyspnea</td>
</tr>
<tr>
<td>Myocarditis</td>
<td>Same as AMI or pericarditis</td>
<td>Dyspnea, palpitations, CHF</td>
</tr>
<tr>
<td><strong>RESPIRATORY</strong></td>
<td></td>
<td></td>
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<tr>
<td>Pulmonary embolus</td>
<td>Sharp pleuritic or central ache</td>
<td>Dyspnea, cough, hemoptysis, leg swelling, risk factors</td>
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<tr>
<td>Pneumothorax</td>
<td>Sudden, sharp, pleuritic</td>
<td>Dyspnea, cough</td>
</tr>
<tr>
<td>Pneumomediastinum</td>
<td>Variable</td>
<td>Risks: cocaine, COPD,iatrogenic procedures</td>
</tr>
<tr>
<td>Pneumonia</td>
<td>Sharp, pleuritic</td>
<td>Cough, fever, dyspnea</td>
</tr>
<tr>
<td>Pleuritis</td>
<td>Sharp, pleuritic</td>
<td>Cough, fever, fever, dyspnea</td>
</tr>
<tr>
<td>Mediastinitis</td>
<td>Variable</td>
<td>Fever, dyspnea, sepsis</td>
</tr>
<tr>
<td>Tumor</td>
<td>Chronic, variable</td>
<td>Weight loss</td>
</tr>
<tr>
<td><strong>ESOPHAGEAL</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Rupture: Boerhaave</td>
<td>Sudden, severe</td>
<td>Vomiting, hematemesis</td>
</tr>
<tr>
<td>Esophageal spasm</td>
<td>Similar to AMI</td>
<td>Reflux, nausea</td>
</tr>
<tr>
<td>Reflux esophagitis</td>
<td>Burning; worse supine</td>
<td>Reflux, nausea, sore throat</td>
</tr>
<tr>
<td><strong>ABDOMINAL DISORDERS</strong></td>
<td></td>
<td></td>
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<tr>
<td>Gastrointestinal</td>
<td>Constant, related to food</td>
<td>Vomiting, abdominal pain, GI bleeding</td>
</tr>
<tr>
<td>Cholecystitis</td>
<td>Constant or colicky</td>
<td>Vomiting, abdominal pain, jaundice, fever</td>
</tr>
<tr>
<td>Ruptured ectopic pregnancy</td>
<td>Sharp, pleuritic</td>
<td>Abdominal pain, vaginal bleeding, shoulder pain</td>
</tr>
<tr>
<td><strong>MUSCULOSKELETAL</strong></td>
<td></td>
<td></td>
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<tr>
<td>Ruptured cervical disc</td>
<td>Pain on neck movement</td>
<td>Neurologic signs, pain referred in root distribution</td>
</tr>
<tr>
<td>Costochondritis</td>
<td>Sharp, pleuritic</td>
<td>Localized tenderness and inflammation</td>
</tr>
<tr>
<td>Herpes zoster</td>
<td>Burning, lancinating</td>
<td>Dermatomal distribution of pain, rash, paresthesias</td>
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<tr>
<td>Postherpetic neuralgia</td>
<td>Burning, lancinating</td>
<td>History of zoster</td>
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</tbody>
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midline epigastric discomfort usually described as indigestion or burning. It lasts minutes to hours, and is worse after eating and lying down. It is associated with belching but not shortness of breath, and tends to be chronic; it may be associated with an acid taste.

Relief with antacids does not reliably rule out pain from cardiac ischemia. Esophageal rupture produces pain in the anterior chest, back, or epigasstium. Rupture occurs in the setting of increased barotrauma from retching or prolonged vomiting or coughing. Vomiting that occurs before the pain suggests esophageal rupture, not AMI (14). Dysphagia and occasionally hemoptysis are the initial symptoms. Life-threatening medias-tinitis and sepsis develop if esophageal rupture goes untreated.

**DIFFERENTIAL DIAGNOSIS**

The following immediately or potentially life-threatening diseases of the thoracic organ systems must be considered in every patient who presents to the emergency department with chest pain.

**Acute Myocardial Infarction and Ischemia**

Occlusion of a coronary vessel resulting in death of heart muscle can present as a catastrophic condition, with malignant dysrhythmias, pump failure, or myocardial rupture, or as a completely compensated minimally painful state. It is by far the most common chest pain emergency, accounting for 40 of 1,000 hospital admissions (2). Although the Framingham study risk factors of smoking, hypertension, diabetes, family history, and elevated cholesterol further increase the likelihood of developing AMI or angina over a period of years, they are not helpful in excluding or confirming the diagnosis in the acute care setting. Nevertheless, the physician should consider, for medicolegal purposes, the presence of risk factors in all patients with chest pain. The symptoms of coronary vasospasm may be very similar to those of AMI or angina due to other disease.

Emergency physicians frequently evaluate patients with pain associated with recent cocaine use (9,24). Such patients commonly have vasospasm, and indeed have a lower confirmed rate of myocardial infarction than non-cocaine-related admissions (4). Vasospastic ischemic pain is more common than AMI. In patients with cocaine-associated chest pain, even in those without ischemia, ST segment elevation is present much of the time due to repolarization abnormalities (9). Unless the presentation of AMI is classic, these abnormal baseline findings make it helpful to have an old electrocardiogram (ECG) without ST elevation, or serial ECGs showing evolving ST elevation in the emergency department, before giving thrombolytic therapy. Cardiac catheterization is the optimal diagnostic and potentially therapeutic maneuver.

In AMI, thrombolytic therapy, percutaneous coronary interventions, adjunctive treatment with antithrombotic and antplatelet agents, and treatment in the coronary care unit have significantly lowered case fatality and reinfarction rates, making it imperative to diagnose an acute coronary syndrome in the
emergency department. Unstable angina is just as important to recognize, because 10% to 20% of confirmed cases proceed to infarction. Heparin, glycoprotein IIb/IIIa and ACE inhibitors, and vasodilators, or more invasive procedures such as coronary revascularization, may be required to treat recurrent pain or associated heart failure or shock (see Chapter 41, Acute Coronary Syndromes).

**Dissection of the Thoracic Aorta**

This disruption in the intimal layer of the aorta allows blood to track within the media. Aortic rupture is an ever-present threat. Untreated dissection has a mortality rate of 90%; fortunately, it is responsible for less than 1 in 1,000 hospital admissions (2). Progression of the dissection can cause severe organ damage to the spine (sometimes misclassified as stroke), brain, kidneys, bowel, and heart, including AMI and cardiac tamponade. The patient must be carefully examined for evidence of these complications. Hypertension is the most common risk factor, although younger patients with Marfan or Ehlers-Danlos syndromes may have dissection due to cystic medial necrosis (see Chapter 55, Thoracic Aortic Dissections and Aneurysms).

**Pulmonary Embolism**

Pulmonary embolism is an obstruction of the pulmonary arterial system due to clot, which usually embolizes from deep femoral or pelvic veins. In addition to dyspnea, pleuritic pain, and hemoptysis, it can produce acute respiratory failure or right-sided heart failure, as evidenced by cyanosis, hypotension, increased jugular venous pressure, and a loud S2 or right heart gallop. The hospitalization rate is 2 in 1,000 (2). Mortality from PE itself is uncommon and is usually due to recurrent embolism. Recognition of such predisposing conditions as immobilization, recent surgery, hypercoagulability, and low-flow circulatory states is critical to raising the clinician's diagnostic suspicion. A high degree of suspicion is especially important, because PE often presents with subtle, nonspecific findings (see Chapter 38, Pulmonary Embolism).

**Tension Pneumothorax**

Tension pneumothorax occurs when air escapes from the lung into the thoracic cavity, shifting the mediastinum to one side and compromising right-sided heart filling. Dyspnea, diaphoresis, tachycardia, tachypnea, and hypotension commonly result. Jugular vein distention, tracheal deviation, decreased breath sounds, and percussion tympany are noted on physical examination. Pneumothorax may be spontaneous or secondary to trauma, infection (such as tuberculosis and AIDS), or the rupture of emphysematous blebs. Patients with tension pneumothorax require immediate needle thoracostomy and subsequent placement of a chest tube (see Chapter 37, Spontaneous Pneumothorax and Pneumomediastinum and Chapter 183, Penetrating Chest Trauma).

**Esophageal Rupture**

Esophageal rupture can occur in the cervical esophagus, but it usually occurs in the distal esophagus. Conditions associated with retching, vomiting, and coughing, such as ethanol abuse, hyperemesis gravidarum, and status asthmaticus, predispose patients to this rare but important disease (21)(see Chapter 58, Esophageal Disease).

**DIAGNOSTIC APPROACH**

After verifying or establishing initial patient stability, the diagnostic approach proceeds from a consideration of age, sex, specific clinical presentation, and pre-existing conditions or risk factors for the specific emergency diagnoses. The history and physical examination are essential for differentiating these conditions and detecting their complications.

The chest x-ray is key to the detection of pneumothorax, heart failure, pneumonia, and a widened mediastinum, as seen in aortic dissection. Mediastinal or subcutaneous air can be a clue to devastating conditions such as mediastinitis or esophageal rupture. Diagnosis of certain entities may require more advanced imaging modalities, such as CT scanning, MRI, angiography, or echocardiography. Transesophageal echocardiography is sensitive, specific, and practical for the emergency department diagnosis of aortic dissection (11).

Diagnosing PE continues to be clinically challenging, as ECG, arterial blood gas, and chest radiography are all nondiagnostic tests. Arterial blood gas studies are useful to assess the severity of hypoxemia, but a normal PaO2 is present about 10% of the time in the patient with PE. The fundamental approach is to risk-stratify patients into high, medium, and low probabilities of disease (3,25). Clinical probability then guides test selection. An assay for d-dimers is a sensitive test, and thus can serve to exclude the diagnosis in patients with low clinical probability of disease (3). For those with high or moderate probability, imaging with thin-slice spiral CT is overtaking V/Q scanning as the confirmatory test of choice (22). However, a negative V/Q scan virtually excludes pulmonary emboli whereas a negative spiral CT does not exclude the presence of smaller, subsegmental clots. Optimal evaluations are guided by risk stratification and local availability of specific tests (3,8,25).

Bedside risk stratification is also the fundamental approach to the diagnosis of acute coronary syndromes. The history, physical exam, and 12-lead ECG are the basis for dividing patients into high and low probability of disease (6,18). In the patient with chest pain or other suggestive symptoms of cardiac ischemia, ST-segment deviation (elevation or depression, or left bundle branch block) or positive troponin testing indicates a high-risk patient.

The ECG is particularly instructive. Regional ST elevation of 1 mm (0.1 mV) in two leads is the indication for coronary reperfusion therapy, and assessment of -segment depression in leads opposite (i.e., reciprocal) to the ST-segment elevation is helpful in confirming subtle ST elevation. Serial ECGs are useful with recurrent pain in higher risk patients to detect AMI evolution or reperfusion (5). The ECG pattern can also provide information favoring other elements of the differential diagnosis. Diffuse ST-segment elevation with associated PR depression (most frequent in lead II) suggests pericarditis; low-voltage or electrical alternans suggests an effusion. Anterior T-wave inversions can be present in myocardial ischemia or pulmonary embolism (17). The ECG may reveal ischemia or an infarction pattern in the inferior leads in patients with aortic dissection, as the right coronary artery may be involved in the dissection. The most common ECG finding in pulmonary embolism is sinus tachycardia, followed by right precordial T-wave inversion, a strain pattern (17). The S1Q3T3 pattern has greater specificity, but is uncommon in pulmonary embolism.

The emergency department ECG is also a simple prognostic indicator for in-hospital complications. In stable, pain-free patients who are being admitted to rule out AMI, an ECG without ST-segment changes, Q waves, T-wave inversion, or left bundle-branch block is associated with a very low (less than 1%) incidence of life-threatening complications. When an ST-segment elevation of 0.1 mV is present in the right ventricular lead V4R.
in the setting of inferior AMI, suggesting coincident right ventricular infarction, complication rates are higher than when it is absent (26).

In addition to bedside risk stratification, formal diagnostic decision tools, such as the algorithm for risk assessment in AMI (6) or a computer-generated numerical probability for acute ischemia (18), have been developed and validated. They are not yet in widespread clinical use. For prognosis, a formal algorithm developed by Goldman and associates can predict short-term complication rates using ECG findings, presence of heart failure, low blood pressure, and worsening angina (7).

The tools and technologies are now well established for the relatively rapid diagnostic assessment of acute cardiac ischemia (15,16,23,24,27). For low probability patients, in the setting of an in-hospital or short stay ED observation bed, serial ECGs (5) followed by serial cardiac markers can confirm or exclude myocardial necrosis (15). Further assessment with provocative testing, such as stress echocardiography or stress nuclear imaging complete the work-up to exclude an acute coronary syndrome (23). Such testing also provides valuable prognostic information (4,7,15,23,25,26). Newer cardiac biomarkers not based on cardiac muscle death, such as an albumin-based assay for coronary ischemia, aim to be the “holy grail” of an early marker of ischemia rather than just necrosis; however, such an assay has not been validated (19). It is now widely accepted by emergency physicians and their in-hospital colleagues that a large portion of chest pain patients can only be evaluated by serial rather than cross-sectional testing. This realization has likely contributed to decreased rates of “missed” ischemia and improved patient care.

CRITICAL INTERVENTIONS

All patients with acute chest pain should have a rapid (≤10 minutes) ECG and then continuous cardiac ECG monitoring, chest radiography, intravenous access, and oxygen. Aspirin, nitroglycerin, and beta-blockers are indicated for suspected ischemic pain. Most patients with unstable angina should also receive low-molecular weight or unfractionated heparin. Patients with MI and high risk ECGs (ST-segment elevation, left or right bundle-branch block, ST-depression with ongoing ischemic pain) should have an immediate cardiology consultation. Patients with suspected pulmonary embolism receive a similar diagnostic approach, but oxygen and low-molecular weight or unfractionated heparin are the initial treatments. Patients with aortic dissection must have immediate blood pressure control with both a beta-blocking agent and a vasodilator, and no anticoagulation. Tension pneumothorax with cardiovascular compromise (absent breath sounds, distended neck veins, tracheal deviation) is a clinical diagnosis and requires immediate decompression with a 16-gauge angio-catheter followed by chest tube insertion and hospitalization.

DISPOSITION

All of the life-threatening elements in the differential diagnosis of chest pain must be excluded prior to discharge from the ED. Chest pain with features typical for active acute ischemia requires the initiation of anti-arrhythmic and anti-arrhythmia therapies in the ED with continuation in the hospital. Cardiac consultation and coronary care unit admission is indicated for all such patients. Transfer to percutaneous transluminal coronary angioplasty (PTCA) capable institutions should be considered for patients in need of reperfusion (ST-segment elevation or left bundle-branch block) but ineligible for thrombolytic therapy, and for those with active ischemic pain. Patients with lower probabilities of ischemia and complications can be safely and effectively ruled out in an emergency department or in-hospital observation unit. This can be done over a 6- to 9-hour period with serial biomarkers, followed by a cardiovascular stress test prior to discharge or within 2 to 3 days of discharge. Patients with pulmonary embolism may need ICU admission if they are hemodynamically unstable or have refractory hypoxemia; for smaller emboli, admission to a telemetry bed or the medical floor is appropriate. Patients with pericarditis should undergo echocardiography if pericardial effusion or tamponade are suspected, and may require a period of observation in the hospital. However, stable patients with pericarditis can be discharged from the ED.

COMMON PITFALLS

✓ Ordering a single cardiac biomarker or enzyme to exclude myocardial infarction
✓ Failure to order serial ECGs in patients with a negative ECG and a history suggestive of an acute coronary syncope
✓ Believing that right bundle-branch block prevents detection of ST-segment elevation
✓ Believing that therapeutic maneuvers of sublingual NTG or antacids have diagnostic value
✓ Failure to consider the diagnosis of esophageal rupture and palpate the soft tissues of the neck for subcutaneous emphysema
✓ Failure to consider the diagnosis of aortic dissection in patients with anterior or posterior chest pain
✓ Believing that pain relief with sublingual nitroglycerin excludes aortic dissection
✓ Failure to examine the chest x-ray for the absence of pulmonary vascular markings extending to the parietal pleura

References